AMENDMENTS TO THE CLAIMS:

1. (Currently Amended) A Method of correlating the ability of a cell expressing FcαRI to bind IgA and cellular susceptibility to a disease, said method comprising:

identifying a FcαRI gentotype of said cell for FcαRI alleles selected from the group consisting of: FcαRIa 87R/87R, FcαRIA 92D/92N, FcαRIa 132F/132L, FαRI 245P/245L and FcαRI 248S/248G;

quantifying IgA binding by said cell expressing said FcαRI gentotype; and

comparing IgA binding by said cell and IgA binding by a second cell, said second cell expressing a second FcαRI genotype, wherein correlation of the ability of said cell to bind IgA and celluar susceptibility to disease is indicated by a difference in IgA binding detected by said comparing.

2. (Original) The method of claim 1 wherein said FcαRI genotype differs from said second FcαRI genotype by a point mutation.

3. (Original) The method of claim 2 wherein said point mutation is a silent mutation.

4. (Original) The method of claim 2 wherein said point mutation is a frame shift mutation.

5. (Original) The method of claim 2 wherein said point mutation is a missense mutation.

6. (Original) The method of claim 3 wherein said silent mutation is in codon 87 of said FcαRI genotype.

7. (Original) The method of claim 3 wherein said silent mutation is in codon 92 of said FcαRI genotype.

8. (Original) The method of claim 5 wherein said missense mutation is at codon 132 of said FcαRI genotype.

9. (Original) The method of claim 5 wherein said missense mutation is at codon 245 of said FcαRI genotype.

10. (Original) The method of claim 5 wherein said missense mutation is at codon 248 of said FcαRI genotype.

11. (Original) The method of claim 1 wherein said disease is selected from the group consisting of: periodontal disease, cancer, viral infection, bacterial infection, systemic lupus erythematosus, systemic vasculitis, IgA nephropathy, rheumatoid arthritis, systemic sclerosis, dermatomyositis, Hashimoto's thyroiditis, inflammatory bowel disease and Sjogren's syndrome.

12. (Original) The method of claim 1 wherein said cell is selected from the group consisting of: a neutrophil, a monocyte, a myeloid cell, and a mucus secreting cell.

13. (Currently Amended) A method for determining FcαRI alleles specific to an individual human, said method comprising: genotyping DNA encoding FcαRI for a polymorphism selected from the group consisting of: FcαRIa 87R/87R, FcαRIA 92D/92N, FcαRIa 132F/132L, FαRI 245P/245L and FcαRI 248S/248G, said DNA being obtained from said individual human.

14. (Original) The method of claim 13 wherein said polymorphism affects IgA binding by a FcαRI receptor.

15. (Original) The method of claim 13 wherein said polymorphism affects signal transduction.

16. (Original) The method of claim 13 wherein said polymorphism is a single nucleotide polymorphism.

17. (Original) The method of claim 13 wherein said polymorphism is a microsatellite polymorphism.

18. (Original) The method of claim 13 wherein said polymorphism is a splice isoform.

19. (Original) The method of claim 13 wherein said polymorphism is in the glycosylation sites of FcαRI.

20. (Original) The method of claim 13 wherein genotyping utilizes PCR typing with a sequence specific primer for a polymorphic exon.

21. (Original) The method of claim 20 wherein said primer is selected from the group consisting of those shown in Example 4.

Claims 22-25 (Withdrawn)

26. (Currently Amended) A method of prognosticating a human immunoresponse to a disease, said method comprising:

establishing a correlation between a FcαRI genotype for a FcαRI alleles selected from the group consisting of: FcαRIa 87R/87R, FcαRIA 92D/92N, FcαRIa 132F/132L, FαRI 245P/245L and FcαRI 248S/248G and clinical outcome of said disease;

genotyping a patient for FcαRI to yield a patient FcαRI genotype;

comparing said FcαRI genotype with said patient genotype; and

determining clinical outcome for said patient based on said patient genotype, wherein determining said clinical outcome is indicative of a human immunoresponse to a disease.

27. (Original) The method of claim 26 wherein genotyping utilizes PCR typing with a sequence specific primer for a polymorphic exon.

28. (Original) The method of claim 27 wherein said primer is selected from the group consisting of those shown in SEQ ID Nos. 1, 2, 3 and 4.

29. (Original) The method of claim 26 wherein genotyping comprises purifying FcαRI expressing cells from said patient; extracting nucleic acids from said cells; and determining whether the nucleic acid encodes a predetermined polymorphic FcαRI nucleic acid sequence.

30. (Original) The method of claim 29 wherein the nucleic acid is selected from the group consisting of: RNA and DNA.

Claims 31-33 (Cancelled)

34. (Original) A commercial package comprising reagents for identifying single nucleotide polymorphisms in a FcαRI genotype or phenotype together with instructions for use thereof as a test to identify individual susceptibility to a disease.

Claim 35 (Cancelled)

36. (Currently Amended) A method for correlating response of a cell expressing FcαRI to binding FcαRI ligand and cellular susceptibility to a disease, said method comprising:

identifying a FcαRI genotype of the cell for FcαRI allele selected from the group consisting of: FcαRIa 87R/87R, FcαRIA 92D/92N, FcαRIa 132F/132L, FαRI 245P/245L and FcαRI 248S/248G;

quantifying response to binding FcαRI ligand by the cell expressing the FcαRI genotype;

comparing response to binding FcαRI ligand by the cell and response to binding FcαRI ligand by a second cell, the second cell expressing a second FcαRI genotype, wherein correlation of the response of a cell to binding FcαRI ligand and cellular susceptibility to disease is indicated by a difference in response detected by the comparing.

37. (Previously Presented) The method of claim 36 wherein the FcαRI genotype differs from the second FcαRI genotype by a point mutation.

38. (Previously Presented) The method of claim 37 wherein the point mutation is a frame shift mutation.

39. (Previously Presented) The method of claim 37 wherein the point mutation is a missense mutation.

40 (Previously Presented) The method of claim 37 wherein the point mutation alters a phosphorylation site of FcαRI.

41. (Previously Presented) The method of claim 40 wherein the phosphorylation site is a casein kinase I phosphorylation site.

42. (Previously Presented) The method of claim 36 wherein the response is activation of an enzyme.

43. (Previously Presented) The method of claim 36 wherein the response is induction of phagocytosis.

44. (Previously Presented) The method of claim 36 wherein the response is induction of oxidative burst.

45. (Previously Presented) The method of claim 36 wherein the response is induction of cytokine production.

46. (Previously Presented) The method of claim 36 wherein the response is release of collagenase.